

Exercise walk-through

1.2

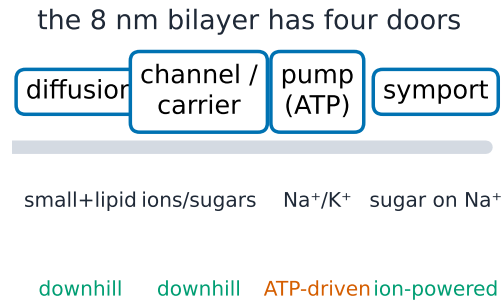
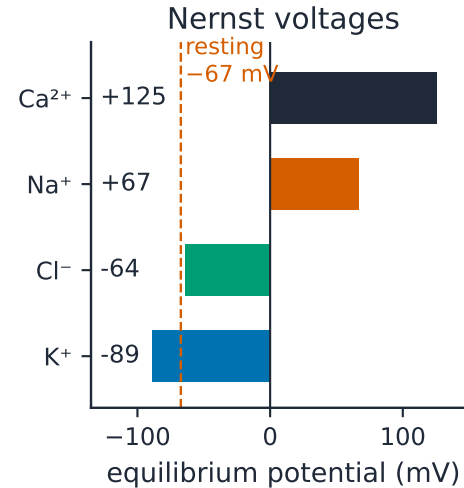
Membranes and transport

University Medicine

Membranes: four doors and a voltage

- 1 **Voltages** K^+ -89 mV, Cl^- -64 mV, Na^+ $+67$ mV, Ca^{2+} $+126$ mV - the **resting potential** sits a hair above E_K because P_K dominates.
- 2 **Emergency** Extracellular K^+ doubles $5 \rightarrow 10$ mM: E_K shifts $+18.5$ mV $= 61.5 \cdot \log_{10}(2)$ - the hyperkalaemia mechanism is one logarithm.
- 3 **Pump** 3 Na^+ out, 2 K^+ in, 1 ATP: the pump holds the gradients the leak channels spend; V_m rests just above E_K .

The four doors



Four doors cross the 8 nm bilayer; the Nernst bars show why K⁺ sets the resting voltage.

Exercises 2.1-2.3

2.1 Compute E_K for $[K]_o = 15 \text{ mM}$, $[K]_i = 140 \text{ mM}$. [3]

2.2 Which door does glucose take into an erythrocyte, and why not simple diffusion? [3]

2.3 Why does doubling $[K]_o$ shift E_K by exactly $61.5 \log_{10} 2 \text{ mV}$? [3]

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2.1

- $61.5 \log_{10}(15/140) = 61.5 \times (-0.970) = -59.7 \text{ mV}$.
-

2.2 Which door does glucose take into an erythrocyte, and why not simple diffusion? [3]

2.2

- Facilitated diffusion via the GLUT carrier: glucose is hydrophilic, so the bilayer alone is impassable; movement is downhill, no ATP.
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2.3 Why does doubling $[K]_o$ shift E_K by exactly $61.5 \log_{10} 2 \text{ mV}$? [3]

2.3

- $E_K \propto \log_{10}([out]/[in])$; doubling $[out]$ adds $\log_{10} 2$ inside the log: $+18.5 \text{ mV}$.

Exercise 3.1

3.1 A (fictional) patient's serum K^+ rises $5 \rightarrow 10$ mM.

Compute the shift in E_K and the new value. Explain why the resting potential depolarises and why this is a cardiac emergency. State what the Na^+/K^+ pump does to the gradients and its stoichiometry. [8]

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3.1

- Shift $61.5 \log_{10} 2 = +18.5$ mV; new $E_K = -89.0 + 18.5 = -70.5$ mV ✓.
- V_m tracks E_K (P_{K^+} dominates), so depolarisation.
- Cardiac: depolarised myocytes sit closer to threshold – arrhythmia risk.
- Pump: 3 Na^+ out, 2 K^+ in, 1 ATP – it maintains the gradients but cannot by itself fix an extracellular load.

Exercise 3.2

3.2 $\Delta F508$ cystic fibrosis (fictional case used as mechanism).

Name the door class affected. Trace the mechanism from misfolded channel to dehydrated airway surface. Explain why salt and water follow the trapped Cl^- (osmosis, 290 mOsm). State why small molecules that hold the fold open are a therapy rationale. [8]

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3.2

- Door: a Cl^- channel (facilitated diffusion class).
- Misfolded $\rightarrow$ retained in the ER $\rightarrow$ never reaches the membrane $\rightarrow$ Cl^- cannot leave.
- Cl^- trapped $\rightarrow$ salt accumulates luminal-side $\rightarrow$ water follows osmotically (290 mOsm gradients equalise) $\rightarrow$ surface liquid dehydrates $\rightarrow$ mucus cannot clear.
- Therapy: folding correctors hold the channel open through processing, restoring some Cl^- flow.